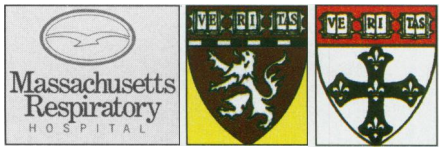
GRAND ROUNDS in Environmental Medicine



MASSACHUSETTS RESPIRATORY HOSPITAL,
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A Retired Shipyard Worker with Rapidly Progressive Pulmonary Interstitial Fibrosis

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We present a case of progressive interstitial fibrosis in a retired shipyard worker who was exposed to asbestos during the postwar era of the late 1940s and 1950s, when asbestos exposures in the workplace were not regulated. Forty years later, at 63 years of age, the patient presented with restrictive lung disease. The patient was diagnosed with asbestos-related pleural disease and parenchymal asbestosis. He remained stable for the next 7 years, but then he began to manifest rapid clinical progression, which raised the possibility of an unusual variant of asbestosis, a concomitant interstitial process, or an unrelated disease. Lung biopsy was not undertaken because of the patient's low pulmonary reserve and limited treatment options. An empiric trial of oral steroids was initiated, but his pulmonary status continued to deteriorate and he died of pulmonary failure at 72 years of age . Many diseases result in pulmonary interstitial fibrosis. Ideally, open lung biopsy should be performed, but this procedure inevitably causes complications in many patients with end-stage restrictive lung disease. Furthermore, while the presence of asbestos bodies in tissue sections is a sensitive and specific marker of asbestos exposure, neither this finding nor any other charge is a marker indicative of asbestosis or the severity of asbestosis. With the enactment of the Asbestos Standard in the United States, asbestos exposures have been decreasing in this country. However, industries that produce asbestos products and wastes continue to expand in developing countries. Prevention of asbestos-related lung disease should be a global endeavor, and asbestos exposures should be regulated in both developed and developing countries. *Key words:* asbestos, lung, pulmonary interstitial fibrosis. *Environ Health Perspect* 107:321–327 (1999). [Online 17 March 1999]
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Case Presentation

A 63-year old former carpenter was referred in 1988 to the Center for Occupational and Environmental Medicine (COEM), at the Massachusetts Respiratory Hospital, Braintree, Massachusetts, for evaluation of possible asbestos-related lung disease. In 1948 he had begun to work in a shipyard and was involved in the construction, repair, and demolition of vessels. He often worked below deck in enclosed environments, where he was exposed to visible clouds of asbestos dust. Like other workers in the postwar shipbuilding industry, he was not warned that asbestos exposure could be hazardous and he did not wear a respiratory protective device. In 1963 he changed to a desk job and had no further known asbestos exposures. By 1988, the news that his former co-workers at the shipyard were being diagnosed with asbestos-related lung disease prompted him to seek medical screening.

He first consulted a primary care physician. At the time, he was physically very active and denied dyspnea, cough, sputum production, chest pain, palpitations, or fatigue. He had never smoked cigarettes, although he had occasionally smoked cigars and pipes in the past. However, his second wife smoked two to three packs of cigarettes per day at home, and his co-workers at his various workplaces often smoked cigarettes on the job. He was diagnosed with mild diabetes mellitus, which was well controlled with 2.5 mg glipizide/day.

A posteroanterior chest radiograph revealed bilateral pleural plaques and mild diffuse interstitial fibrosis (Fig. 1A). At this point, the patient was referred to this center. Studies of his ventilatory function included forced vital capacity (FVC), forced expiratory volume in 1 sec (FEV_1), total lung capacity (TLC), residual volume (RV), calculated FEV_1/FVC, and a single-breath diffusing capacity for carbon monoxide (DLCO). These tests showed a moderate restrictive defect with a normal diffusing capacity (Table 1). On the basis of his occupational history, chest X-ray findings, and pulmonary function studies, the patient received a diagnosis of asbestos-related pleural plaques and asbestosis.

The patient returned for follow-up at the center every 1–2 years. From 1991 to 1995, the 71-year-old man reported only mild dyspnea on exertion. During a follow-up visit in 1996, however, he complained of a 4–5 month history of shortness of breath while walking his dog and mowing his lawn. On physical examination, he had bibasilar dry "Velcro" rales, but no clubbing or cyanosis and no remarkable findings on cardiovascular examination.

A review of serial chest X rays (Fig. 1A–1C) from 1988, 1992, and 1996 suggested a worsening of his interstitial lung disease. Comparison of high-resolution computed tomography (HRCT) scans obtained in 1991 and 1996 provided firmer evidence that the bilateral interstitial process had progressed from moderate to severe and also yielded additional evidence of pleural plaques (Fig. 2). Moreover, serial pulmonary function studies from 1988, 1992, and 1996 revealed an increase in the patient's restrictive defect, with more rapid declines in his lung volumes between 1992 and 1996 than between 1988 and 1992. His FVC of 2.34 liters (61% of predicted) in 1988 dropped to 1.89 liters (52% of predicted) in 1992, and then to 1.25 liters (35% of predicted) in 1996. His TLC of 3.61 liters (60% predicted) in 1988 dropped to 3.22 liters (55% of

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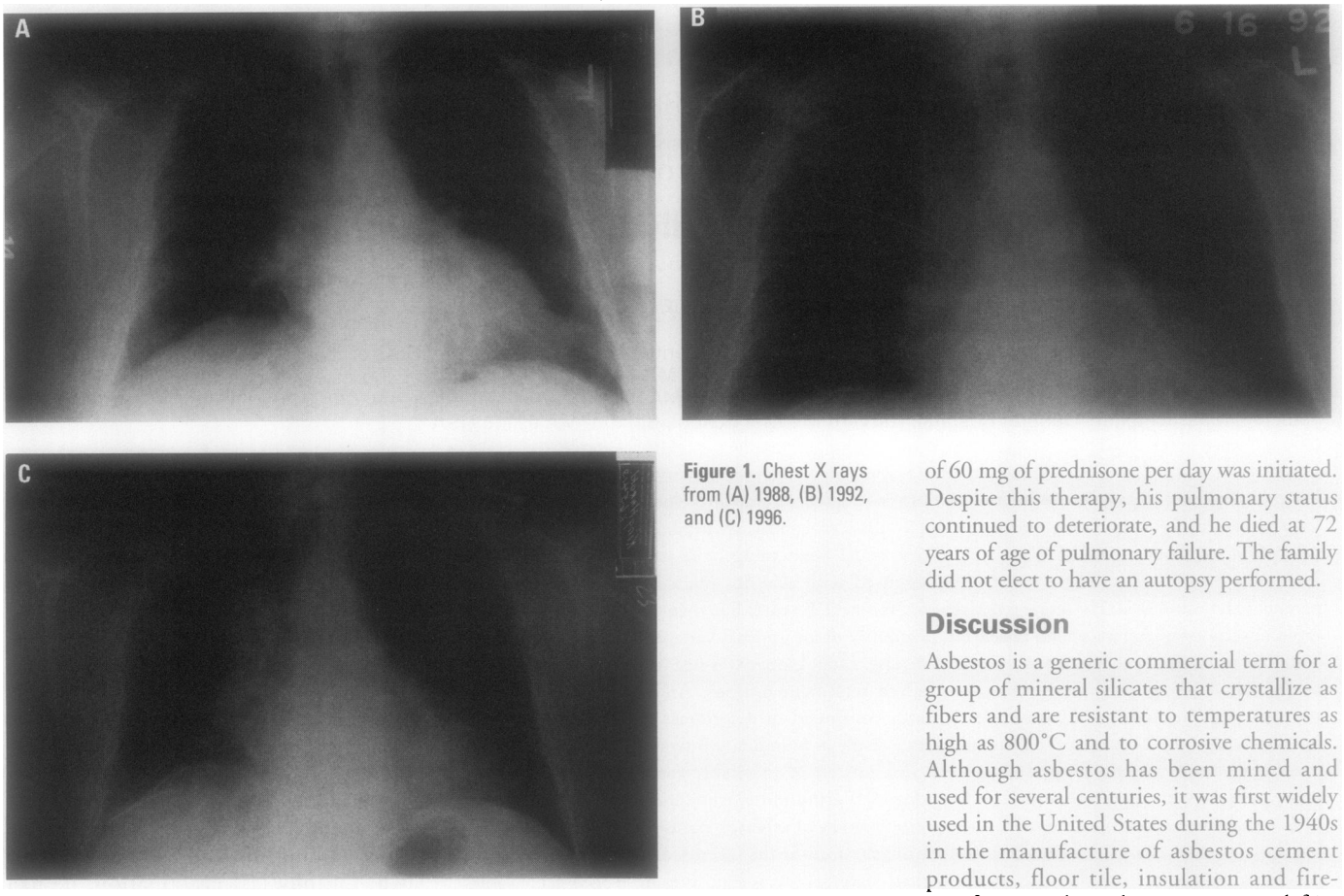

Figure 1. Chest X rays from (A) 1988, (B) 1992, and (C) 1996.

Table 1. Pulmonary function tests

Test	Date	FEV_1 (l)	FVC (l)	FEV_1/FVC (% Pred)	TLC	Single-breath DLCO (% Pred)
Prebronchodilator	1988	1.92 (61)[a]	2.34 (61)	101	3.61 (60)	85
	1992	1.59 (54)	1.89 (52)	105	3.22 (55)	85
	1996	1.03 (37)	1.25 (35)	104	2.33 (40)	59
Postbronchodilator						
	1988	2.04 (65)	2.48 (65)	101		
	1992	1.70 (58)	1.97 (55)	107		
	1996	1.16 (42)	1.30 (37)	113		

Abbreviations: FEV1, forced expiratory volume in 1 sec; FVC, forced vital capacity; TLC, total lung capacity; DLCO, diffusing capacity for carbon monoxide; % Pred, percent of predicted.

[a]Values shown in parentheses are % Pred.

predicted) in 1992 and then to 2.33 liters (40% of predicted) in 1996. Of note, his pulmonary function tests also suggested a mild bronchodilator response (Table 1).

Other studies revealed that the patient's single-breath diffusing capacity was normal at 85% of predicted from 1988 to 1992, but dropped sharply to 59% of predicted in 1995 (Table 1). His arterial blood gas on room gas in 1996 showed a pH of 7.40, a pCO_2 (partial pressure) of 46 mmHg, a pO_2 of 67 mmHg, and an oxygen saturation of 93%. However, when exercise oximetry was conducted as a study of his functional capacity, his oxygen saturation dropped to 88% after he walked 1,100 feet in 6 min. It was evident that the patient's interstitial lung disease was progressing rapidly and that he was becoming hypoxemic.

A workup was initiated for conditions other than asbestosis that could be contributing to a more acute course. No clinical evidence was found to suggest hypersensitivity pneumonitis or systemic diseases associated with pulmonary fibrosis, such as rheumatoid arthritis, collagen vascular disease, or sarcoidosis. Lung biopsy, either open or transbronchial, was not undertaken because of the patient's low pulmonary reserve and limited treatment options. Given the possibility, however small, that he may have had a steroid-responsive condition, an empiric trial of 60 mg of prednisone per day was initiated. Despite this therapy, his pulmonary status continued to deteriorate, and he died at 72 years of age of pulmonary failure. The family did not elect to have an autopsy performed.

Discussion

Asbestos is a generic commercial term for a group of mineral silicates that crystallize as fibers and are resistant to temperatures as high as 800°C and to corrosive chemicals. Although asbestos has been mined and used for several centuries, it was first widely used in the United States during the 1940s in the manufacture of asbestos cement products, floor tile, insulation and fireproofing, textiles, asbestos paper, and friction materials. In this country, the highest level occupational exposures to asbestos have occurred in the repair and demolition of buildings and ships, particularly affecting pipe fitters, boilermakers, and other workers in the building trades (*1*).

The massive shipbuilding effort of World War II placed the largest segment of workers in the United States at risk for developing asbestos-related disease (Fig. 3). Skip construction entailed unusually intense use of insulating materials. The risk was magnified by the relaxation of workplace safety efforts in the wartime economy (*2*). Ships are unusually vulnerable to fire because of their isolation when at sea and their many confined spaces; shipbuilding promotes indirect asbestos exposures because all work takes place within enclosed, poorly ventilated, and unmonitored environments.

Asbestos fibers can be divided into two categories: serpentine and amphibole. Chrysolite, a serpentine fiber, accounts for 90% of globally produced asbestos. Amphibole fibers are of less industrial importance and include crocidolite, amosite, and anthophyllite. Starting in the 1970s, synthetic mineral fibers, such as fiberglass or slag wool, began to replace asbestos. However, asbestos is still used in

the manufacture of brake linings and remains in place as pipe and boiler insulation in thousands of workplaces and homes in the United States. Despite knowledge of effective means for prevention of exposures and institution of stringent standards mandating worker training, exposure monitoring, personal protection, and medical surveillance, asbestos exposures are probably continuing among untrained workers in the United States and throughout the developing world (*3*).

Exposure to asbestos may not be limited to persons who directly handle the material. Indirect occupational exposures may occur when employees work near an area of heavy exposure or alongside heavily exposed workers, e.g., painters or electricians who work near insulation workers in a shipyard. Indirect paraoccupational exposures may also take place in households and neighborhoods when workers carry asbestos dust on their clothes, shoes, hair, toolboxes, and lunch boxes and in their automobiles. During the 1940s and 1950s in the United States, wives of asbestos workers sustained household exposures because they regularly shook out or washed their husbands' work clothes (*4*).

Asbestosis

The four pulmonary conditions caused by asbestos include asbestosis; cancer of the lung parenchyma; mesotheliomas of the pleura, pericardium, and peritoneum; and benign changes of the pleura. The term asbestosis is usually reserved for diffuse interstitial fibrosis of the lung, which is a direct reaction to asbestos exposure. Asbestosis is the prototype of diseases caused by inhalation of mineral fibers and is a form of pneumoconiosis, disease caused by the excessive accumulation of dusts in the lung parenchyma (*5*).

Asbestosis is considered to be a slowly progressive disorder, but the extent to which clinical and exposure parameters among individuals with asbestosis can quantitatively predict death from this disease remains to be clarified. The development of diffuse interstitial fibrosis may relate to the intensity and duration of exposure; at least 10 years of exposure usually occur before the manifestation of disease. As in our patient, the subsequent chronic inflammation, collagen deposition, and scarring seem to progress despite cessation of exposure (*6*).

The etiology of asbestosis unclear, but there are two major hypotheses. The oxygen radical hypothesis states that asbestos-induced production of reactive oxygen species (ROS) may be an important mediator of asbestosis development. When asbestos fibers are inhaled and deposited in the lung parenchyma, alveolar macophages accumulate at the site. During phagocytosis, macrophages secrete hydrogen peroxide. Asbestos fibers may act as intracellular or extracellular substrates for the generation of ROS from hydrogen peroxide molecules, and iron within the fibers or redoxactive iron

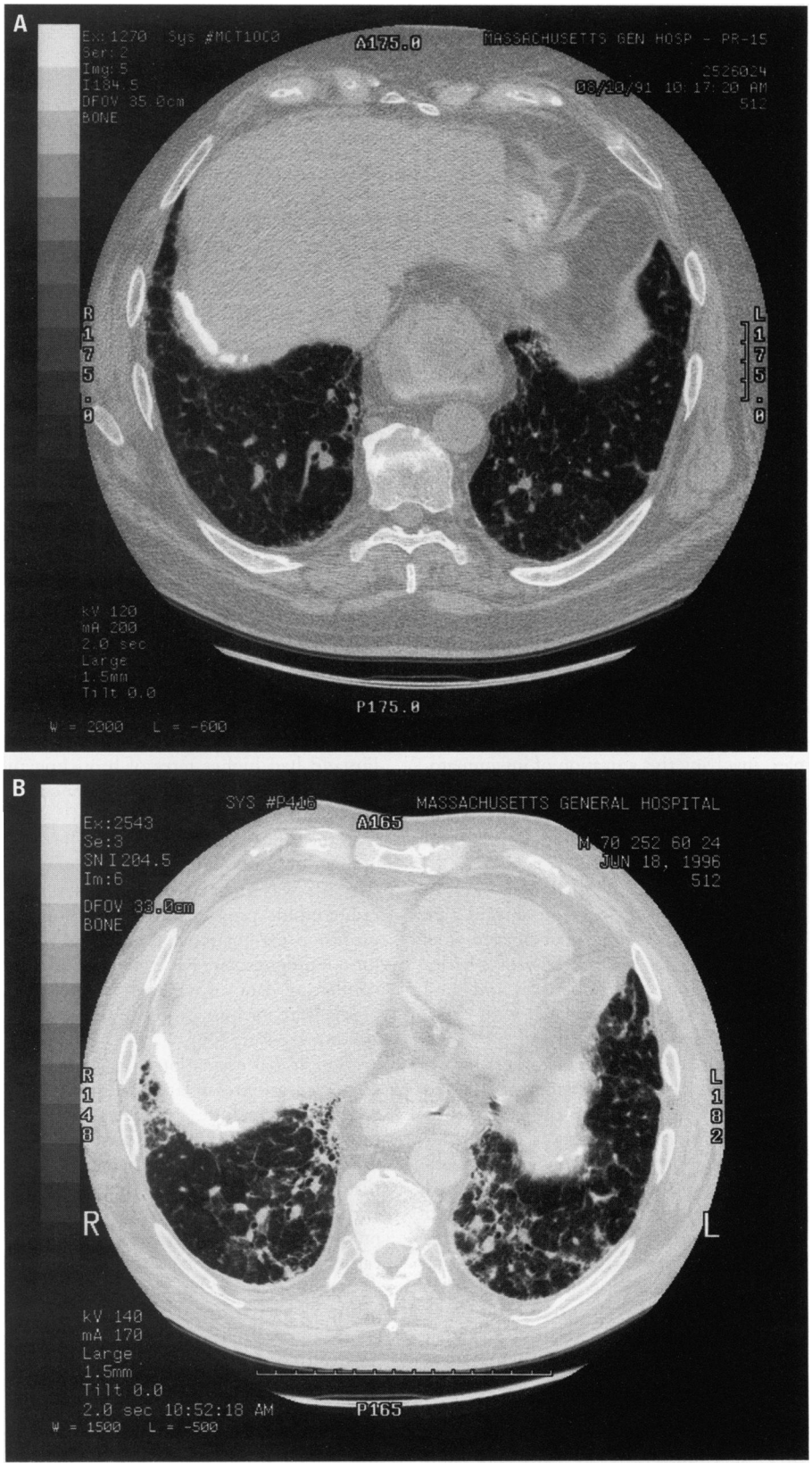

Figure 2. High-resolution computed tomography (HCRT) scans obtained in (A) 1991 and (B) 1996.

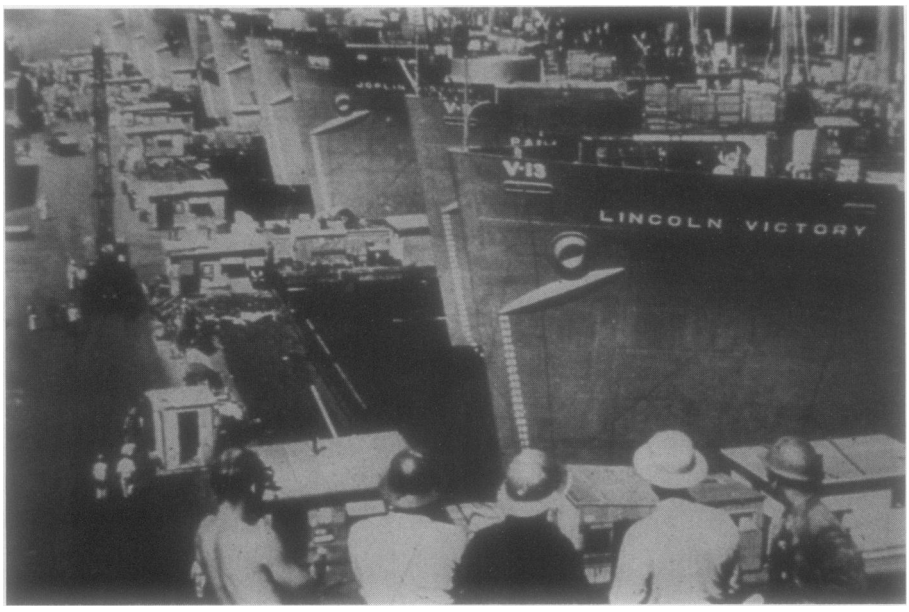

Figure 3. World War II shipyard in San Francisco, California.

associated with or mobilized from the fibers may serve as catalytic agents. ROS in turn may cause DNA damage and cellular toxicity (*7*). The growth factor hypothesis offers an alternative, though not mutually exclusive, explanation. According to this hypothesis, induced cytokines, growth factors, and other proteins act in concert with inflammatory cells to mediate fibrinogenesis (*8*). It seems that the full expression of asbestosis is complex, involving the activation of inflammatory cells, the generation of ROS, and the activation of a multitude of proteins that mediate lung tissue inflammation, injury, and repair at the DNA and cellular levels.

In a necropsy study of textile workers in South Carolina (*9*), lifetime cumulative exposure and total lung fiber burden were strongly correlated with severity of asbestosis. In addition, both asbestosis and lung cancer were strongly correlated with exposure to the long fibers of chrysotile.

It is possible that some workers have a genetically enhanced susceptibility to asbestosis. One approach to the investigation of this area of concern is to look for genetic polymorphisms in the genes encoding for enzymes that repair damage caused by ROS. To date, researchers have focused mainly on metabolic enzymes, such as glutathione *S*-transferase (GST) m and q enzymes, for which homozygous deletions resulting in no enzyme production have been reported to be associated with increased radiographic evidence of asbestosis (*10,11*). Hirvonen et al. (*12*) found that the NAT_2 slow acetylator genotype was more strongly associated with asbestosis than the GST1 deletion. However, many genes and gene products are probably involved in the dynamic processes of damage and repair through complex mechanisms related to environmental exposure, genetic susceptibility, and constitutional factors.

Asbestos-related malignancies may also be associated with asbestosis. Many epidemiologic studies have provided evidence of an elevated incidence of lung cancer among workers with asbestosis. For example, asbestosis patients in a recent study in Finland had higher rates of bronchogenic lung cancer [standardized incidence rate (SIR) = 10] and mesothelioma (SIR = 65) than the general population (*13*). The development of lung cancer among the asbestosis patients was associated with more rapid radiographic progression of small opacity profusion (37 for rapid progressors vs. 4.3 for nonprogressors) (*14*). However, there are conflicting data on whether asbestosis is a prerequisite for lung cancer in the setting of asbestos exposure (*15*).

The initial evaluation of a worker with suspected asbestos-related pulmonary disease should include a clinical history, an exposure history, a smoking history, a physical examination, and anteroposterior chest X rays. The clinical history should focus on cardiopulmonary symptoms. An exploration of cardiac symptoms and functional capacity is important because cardiovascular disease must be excluded in the target age group. The worker with asbestosis may complain of chronic dry cough and dyspnea on exertion, but the degree of symptoms may not correlate well with the extent of disease. Occupational and exposure histories are important in estimation of the duration and extent of asbestos exposure. Smoking history, including passive smoking, is also pertinent to the overall assessment of pulmonary function.

Typically, auscultation of the lungs reveals end-inspiratory basilar "Velcro" rales, the classic finding of pulmonary fibrosis. The examiner should be attentive to findings related to hypoxemia, such as digital clubbing, cyanosis, and tachypnea. The physical examination should be comprehensive because pulmonary fibrosis is nonspecific; many systemic diseases such as rheumatoid arthritis and scleroderma may produce lung findings similar to those of asbestosis.

In cases with a positive history and physical findings, chest radiography should be used for indirect visualization of the lungs. Asbestosis is characterized radiographically by small opacities, most prominent at the lung bases. The size and number (profusion) of these opacities increases with the severity of disease. In severe disease, obscuring of the diaphragmatic and cardiac borders leads to the "shaggy" heart sign. In 1980, the International Labor Organization (ILO) published a classification scheme for the radiographic assessment of pneumoconiosis based on the size and profusion of opacities (*16*). There are limitations to the sensitivity and specificity of chest radiography and ILO profusion scoring. Chest radiography is unreliable in diagnosing and following the progression of interstitial lung disease. The chest radiograph may be normal in the setting of moderate interstitial fibrosis, whereas cigarette smokers tend to have an excess of small, irregular opacities and a relatively high profusion score, even without histologically verified interstitial fibrosis (*17,18*).

Pulmonary function testing is used to augment the clinical database. The earliest changes include diminished vital capacity (VC), reduced DLCO, and an increase in static lung recoil pressures. Subsequent changes include an increase in resting and exercise minute ventilation, arterial desaturation with exercise and later at rest, diminishing TLC, and increased dead space (dead space volume/tidal volume). Large-airway obstruction may be present; this phenomenon is more likely in smokers who have centrilobular emphysema, but is also seen to a lesser extent in nonsmoking asbestos-exposed workers. Small-airway disease may be evident, but is nonspecific in that it may reflect peripheral bronchiolar inflammation and fibrotic narrowing, airway obstruction caused by smoking, or increased lung recoil caused by interstitial fibrosis (*17,19*). Obstructive changes have been observed in asbestosis cases with lower small-opacity profusion scores and prior to more advanced fibrotic stages, in which increased recoil secondary to fibrosis has overcome the effect of decreased airflow (*20*). Agusti

et al. (*21*) studied patterns of gas exchange in response to exercise in asbestosis and idiopathic pulmonary fibrosis; they found more obstruction on exercise. These differences may relate to the underlying morphology of each process: asbestosis may include more airway disease and less pulmonary vascular involvement than other forms of pulmonary fibrosis.

Asbestos exposure as a distinct cause of pulmonary obstruction is controversial because it is difficult to separate smoking as a confounder (*19,22*). In our patient, perhaps exposures to both asbestos and environmental tobacco smoke manifested in obstructive changes. Furthermore, it has been suggested that smoking and asbestos act synergistically to promote both restrictive and obstructive pulmonary disease. For example, Neri et al. (*22*) studied shipyard workers who smoked and were exposed to asbestos: current smokers or ex-smokers with high profusion scores had more obstructive changes, as manifested by lower FEV_1/FVC ratios, than current smokers or ex-smokers with low profusion scores and a shorter duration of smoking. Damage from smoking may decrease the ability to clear inhaled asbestos fibers and increase the permeability of the airway walls, allowing easier penetration by asbestos fibers (*23*).

HRCT scans are considered to be more sensitive than plain films for detecting interstitial fibrosis. There is growing evidence that through increased contrast resolution and axial image display, HRCT can detect both interstitial and pleural disease in advance of clinical symptoms or positive conventional clinical or radiographic studies (*22,24*). In addition, HRCT findings have a greater correlation with the duration of asbestos exposure (*22*), the presence of moderate to severe dyspnea, a high ILO chest X-ray classification, a low FVC, and a high concentration of macrophages and eosinophils in bronchoalveolar lavage fluid (*25*). In combined asbestos and cigarette smoke exposure, HRCT may play a role in distinguishing emphysematous lung destruction from peripheral interstitial changes of asbestosis (*24*). However, expense, time, and the lack of established standards for technique and image interpretation make the use of HRCT inappropriate for initial screening (*21,26*). At present, HRCT is being used in the United States to supplement chest radiography in selected cases.

Asbestosis can also be diagnosed on pathologic grounds. Histologic diagnosis requires the detection of diffuse interstitial fibrosis in the usual pattern and the detection of at least one asbestos body within tissue sections (*27*). An asbestos body (Fig. 4) is an asbestos fiber that penetrates lung tissue and becomes coated with iron and calcium. Ingestion of these fibers by macrophages induces a fibrogenic response through release of growth factors that promote collagen deposition by fibroblasts. In pathologic lung sections, asbestos bodies are best visualized with an iron stain and are also known as ferruginous bodies.

Although there have been a number of studies on asbestosis-associated mortality, our understanding of the risk of death associated with asbestosis remains incomplete. Asbestosis-related mortality has been most closely associated with time since first exposure, intensity of first exposure, and concomitant smoking. A limiting factor in mortality studies, however, is that death due to asbestosis may be attributed to other causes on death certificates (*28*).

Distinguishing Asbestosis from Interstitial Lung Disease of Other Etiologies

It may be difficult to differentiate asbestosis from pulmonary injury resulting from inhalation of other toxic dusts and from other forms of diffuse interstitial pulmonary fibrosis of both the idiopathic and known types. Asbestos-exposed workers are often exposed to many other dusts as well. In shipyards, workers may be exposed to silica, talc, and welding fumes in addition to asbestos. Silicosis can be readily distinguished from asbestosis on a radiographic basis because it typically produces a circumscribed nodular pattern of fibrosis in the upper lobes rather than the irregular linear type of fibrosis that predominates at the lung bases. Bronchiolitis obliterans organizing pneumonia can be differentiated from asbestosis on the basis of the histologic finding of plugs of edematous connective tissue filling the alveolar spaces and alveolar ducts (*17*). Most other diseases that cause diffuse pulmonary fibrosis are not easy to differentiate from asbestosis. Nevertheless, the distinction is important in terms of treatment options. Some interstitial diseases respond to steroids, such as bronchiolitis obliterans organizing pneumonia, desquamative interstitial pneumonia, and sarcoidosis.

"Interstitial lung disease" describes the clinical outcome of more than 100 diseases of both known and unknown causes (*29*).

The known causes include the following:

- Asbestos
- Silica
- Organic dusts
- Fumes, gases
- Drugs (antibiotics and chemotherapy drugs)
- Radiation
- Aspiration pneumonia

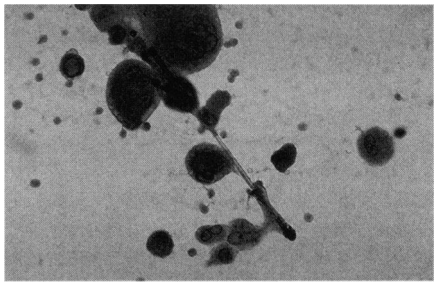

Figure 4. Asbestos body. Reproduced with permission from Edward C. Klatt, Department of Pathology, University of Utah [available: *http://www-medlib.med.utah.edu/WebPath/LUNGHTML/LUNG082.html*].

- Residual of adult respiratory distress syndrome.

The unknown causes include:

- Idiopathic pulmonary fibrosis
- Collagen vascular diseases
- Systemic lupus erythematosus, rheumatoid arthritis, ankylosing spondylitis, systemic sclerosis, Sjogren's syndrome, polymyositis-dermatomyositis
- Pulmonary hemorrhage syndromes
- Goodpasture's syndrome, idiopathic pulmonary hemosiderosis
- Lymphocyte infiltration disorders (lymphocytic interstitial pneumonitis associated with collagen vascular diseases
- Eosinophilic pneumonias
- Lymphangioleiomyomatosis
- Amyloidosis
- Inherited diseases
- Tuberous sclerosis, neurofibromatosis, Niemann-Pick disease, Gaucher's disease, Hermansky-Pudlak syndrome
- Gastrointestinal or liver diseases (Crohn's disease, primary biliary cirrhosis, chronic active hepatitis, ulcerative colitis)
- Graft versus host disease (bone marrow transplantation).

This group of diseases has many common clinical features, including similarities in symptoms, appearance of chest radiographs, alterations in pulmonary physiology, and histologic features. The first response to an initiating event is inflammation in the air spaces and alveolar walls, which causes an acute phase of intraluminal and mural alveolitis. If the exposure is chronic, inflammation may spread to adjacent interstitial and vasculature, producing scarring and distortion of lung tissue along with derangement of gas exchange and ventilatory function. With time, the common end point is diffuse pulmonary fibrosis with resting ventilatory impairment, which will obscure the identity of the initiating event.

The chest radiograph may reveal diffuse reticulonodular markings, particularly at the lung bases. Other subsequently developing radiographic patterns may correlate roughly

with the duration of the disease. An early, hazy, "ground-glass" appearance of the lower-lung fields coincides with the stage of acute alveolitis. Secondarily, curvilinear shadows predominate and may coalesce into nodular infiltrates. Finally, linear opacities are viable in all lung fields in end-stage disease. Another end-stage characteristic is "honeycombing" due to contraction of the lung fields in concert with cystic and bronchiectatic changes. The absence of radiographic changes does not rule out interstitial lung disease, however. Biopsy-proven forms of diffuse interstitial lung disease occur occasionally (~14% of cases) in patients with normal radiographs despite significant exercise intolerance, abnormal pulmonary function tests, and rales (*29*).

Many investigators think that asbestos bodies are the key feature that distinguish asbestosis from other interstitial lung disease. Asbestos bodies in paraffin sections constitute an exquisitely specific marker of significant exposure to asbestos. However, asbestos bodies lack sensitivity as a marker of exposure. In addition, chrysotile fibers accumulate in the lung to a greater extent than amphibole fibers. Therefore, the lack of asbestos bodies may not be informative in the evaluation of amphibole-exposed workers (*27*).

For medicolegal purposes, tissue diagnosis is not necessary for a diagnosis of asbestosis. In 1986, the American Thoracic Society stated that this diagnosis of asbestosis should be based on a careful consideration of all relevant clinical findings, including a reliable history of exposure and an appropriate interval between exposure and detection. Furthermore, the society considered the following clinical criteria to be of value: 1) chest roentgenographic evidence of type "s," "t," "u," small irregular opacifications of a profusion of 1/1 or greater (1/0 is generally considered sufficient, but the committee chose 1/1 because the ILO standard did not contain a 1/0 reference); 2) a restrictive pattern of lung impairment with an FVC below the lower limit of normal; 3) a DLCO below the lower limit of normal; and 4) bilateral late or pan-inspiratory crackles not cleared by cough, at the posterior lung bases (*30*).

In the case we describe here, we proposed an initial diagnosis of asbestosis in 1988 based on occupational history and the presence of pleural plaques, pleural fibrosis, and mild interstitial fibrosis. The progression of clinical interstitial fibrosis that this patient experienced after 1995 seemed to be more rapid than usual for asbestosis, however, suggesting that the patient's disease process represented either a variant of asbestosis or a process unrelated to asbestos exposure. As our case illustrates, obtaining a precise diagnosis can be difficult. Open lung biopsy is the best method for obtaining adequate lung tissue for histologic examination, microbial cultures, and immunofluorescence and electron microscope studies; a transbronchial lung biopsy is usually not sufficient. However, this patient's low respiratory reserve, advanced stage of disease, and limited treatment options dissuaded us from performing either open or transbronchial lung biopsy. Since no theraputic options are currently available for reversing or stabilizing asbestosis and because we thought that this patient might have had a concomitant condition that was steroid responsive, we opted for empiric treatment with corticosteroids.

In the United States, with decreasing exposure to asbestos and promotion of personal protection equipment, the incidence of classical asbestosis will decrease in the next few decades. When physicians encounter a patient with pulmonary fibrosis and a history of asbestos exposure, the possibility of a separate or superimposed process leading to pulmonary fibrosis should be given more serious consideration.

Asbestos: The Public Health Outlook

Today, most patients with asbestos-associated diseases who present to health care facilities were first exposed to asbestos before 1970. Since 1972, the Occupational Safety and Health Administration has passed asbestos standards for general industry (29 CFR § 1910.1001), for shipbuilding (29 CFR § 1915.1001), and for construction (29 CFR § 1926.1101). The current 8-hr time-weighted average permissible exposure limit for asbestos is 0.2 fibers/cm^3 of air, and the short-term excursion limit is 1 fiber/cm^3 of air over a sampling period of 30 min (*31*). The EPA has also regulated asbestos under the Clean Air Act; the Clean Water Act; the Comprehensive Environmental Response, Compensation, and Liability Act; the Food, Drug, and Cosmetic Act; the Resource Conservation and Recovery Act; the Safe Drinking Water Act; the Superfund Amendments and Reauthorization Act; and the Toxic Substances Control Act (*32*).

These regulations have resulted in the reduction of both asbestos use and asbestos exposures in the United States, and asbestos-related disease in the United States is now likely to be a result of past exposures. However, physicians who treat immigrant populations or work in international health settings should be aware that asbestos exposures are on the rise in developing countries. Ironically, hazardous industries, especially those involving substances banned in the United States, are being exported to developing countries. The production of asbestos has been rising in countries such as Brazil, China, and the former Soviet Union. In addition, the importation and use of asbestos in manufacturing has shifted from North America and western Europe to industrializing countries such as Brazil, China, India, Japan, Pakistan, and South Korea (*33–35*). The regulation of asbestos use differs from country to country, and it is likely that many workers are being placed at high risk of asbestos-related diseases because of a lack of stringent regulations, a lack of knowledge about health risks, and potentiating risk factors such as malnutrition and the high prevalence of infectious diseases (*36,37*).

Exposures of the general population to asbestos fibers in air, drinking water, beverages, and foods should also be of concern. Like occupational exposures, environmental exposures to asbestos are accentuated in developing countries. There may be significant asbestos dust in old buildings, particularly buildings that are undergoing reconstruction. Factories that process asbestos or use asbestos in manufacturing may be releasing fibers into the air because of inadequate engineering controls (*38*). The key to preventing asbestos-related lung disease is to control exposures to asbestos in both occupational and environmental settings and in developing as well as developed countries. Physicians may contribute to this effort through accurate diagnosis and disease reporting.

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